

Role of a soy protein Lunasin in A β 42 mediated neurodegeneration in Alzheimer's Disease.

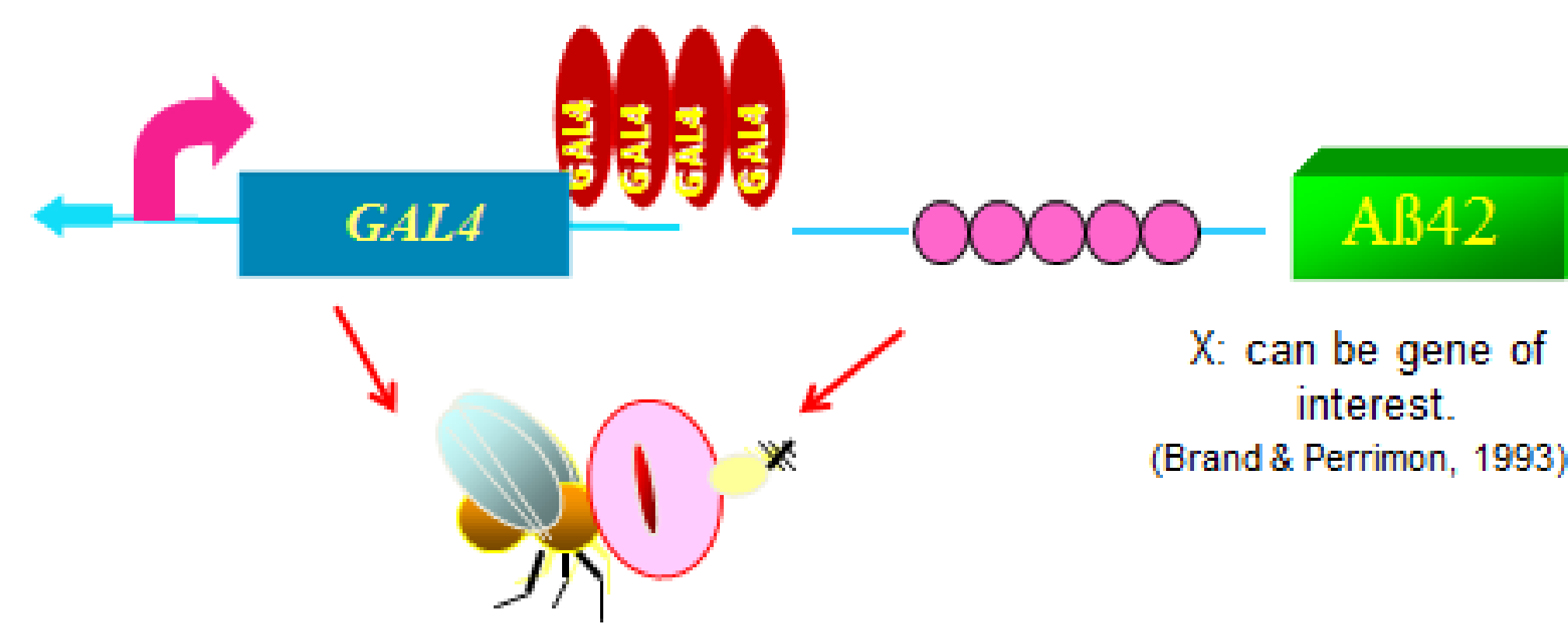
Neil Glenn¹, Ankita Sarkar¹, Gillian Jones², Ajay Srivastava², Madhuri Kango-Singh^{1,3,4} and Amit Singh^{1,3,4}

Department of Biology, University of Dayton, 300 College Park Drive, Dayton, OH; 2) Department of Biology and Biotechnology Center, Western Kentucky University, 1906 College Heights Blvd, Bowling Green, KY 3) Premedical Program, University of Dayton, 300 College Park Drive, Dayton, OH.
4) Center for Tissue Regeneration and Engineering at Dayton (TREND), University of Dayton, Dayton, OH

Abstract

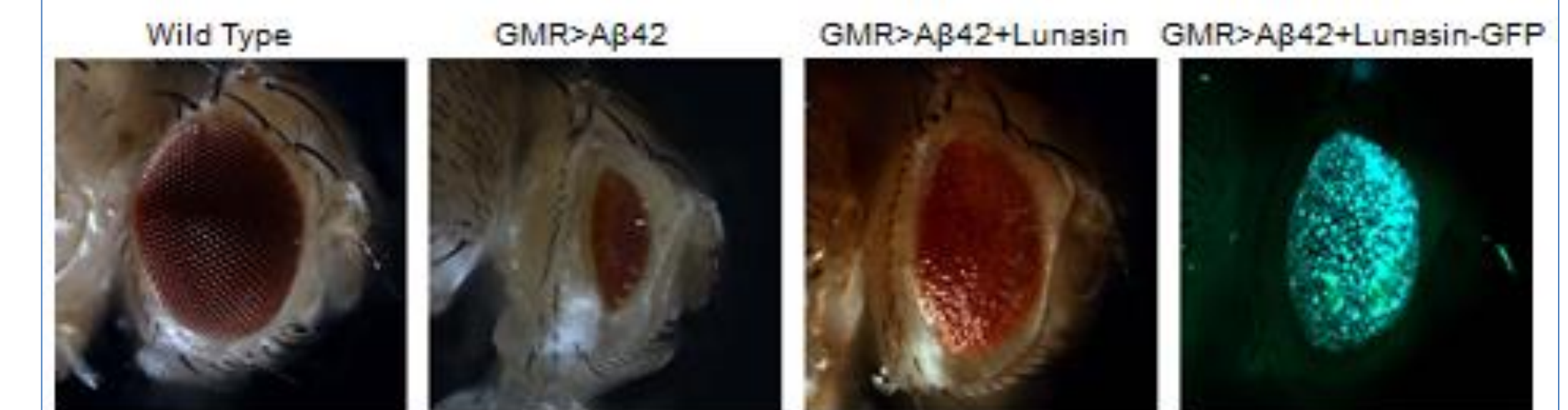
Alzheimer's Disease (AD) is a neurodegenerative disease caused by a number of factors. One of the leading factors behind the onset of AD is the accumulation of amyloid plaques in the brains of affected individuals. These plaques are formed with amyloid precursor protein (APP) is processed incorrectly and cleaved to be 42 amino acids long (A β 42) instead of 40 (A β 40). These two extra amino acids cause the protein to become hydrophobic in nature and form plaques which aggregate around neurons in the brain. This aggregation induces oxidative stress on the neurons which then leads to cell death. Due to the conserved genetic properties of the *Drosophila melanogaster*, fruit fly, visual system with that of humans we have developed a *Drosophila* eye model. In this model the A β 42 protein is misexpressed in the developing photoreceptors of the fly eye which results in extensive cell death of the photoreceptor neurons and produces a highly reduced eye field in the adult fly. Our aim is to understand the function of a soy protein called Lunasin in Alzheimer's disease. It has been shown that Lunasin acts as an anti-inflammatory within the somatic cells. Inflammation is also one of the characteristic of AD. Therefore, we investigated the effects of Lunasin on A β 42 accumulation mediated neurodegeneration. Here we present the findings of our studies.

Gain of-function approach: GAL4/UAS- System



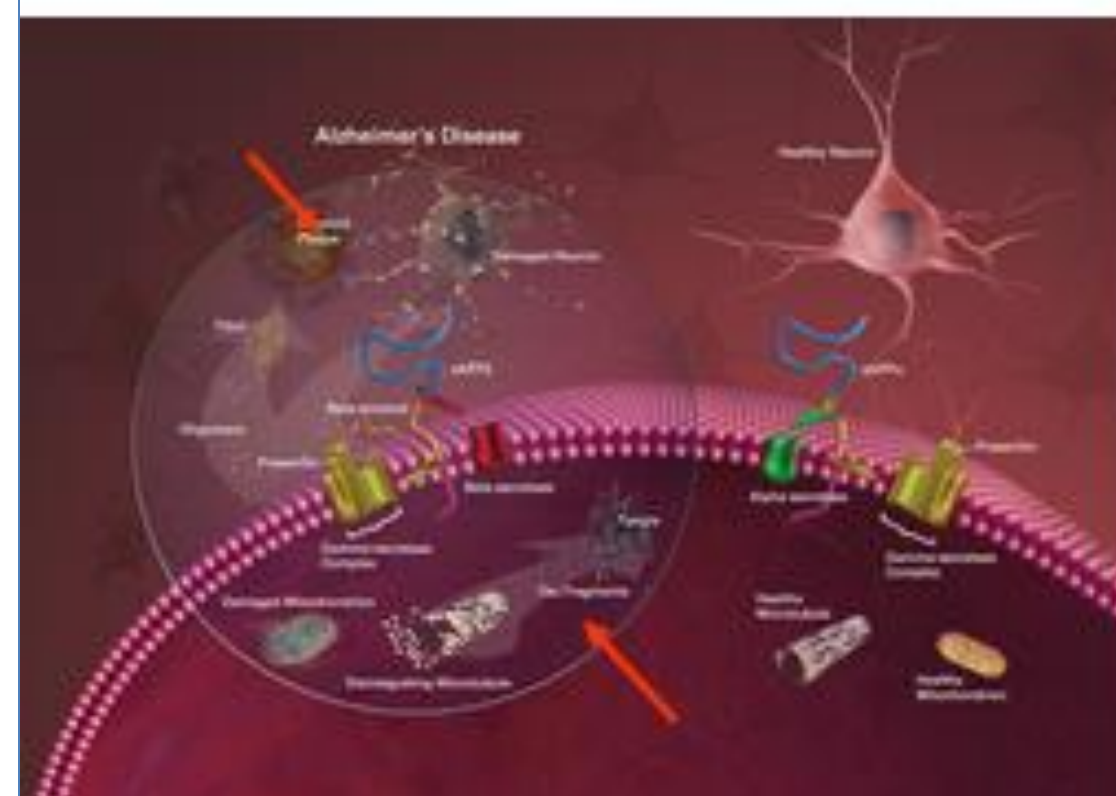
Tare et al., 2011

Lunasin suppresses A β 42 mediated neurodegeneration

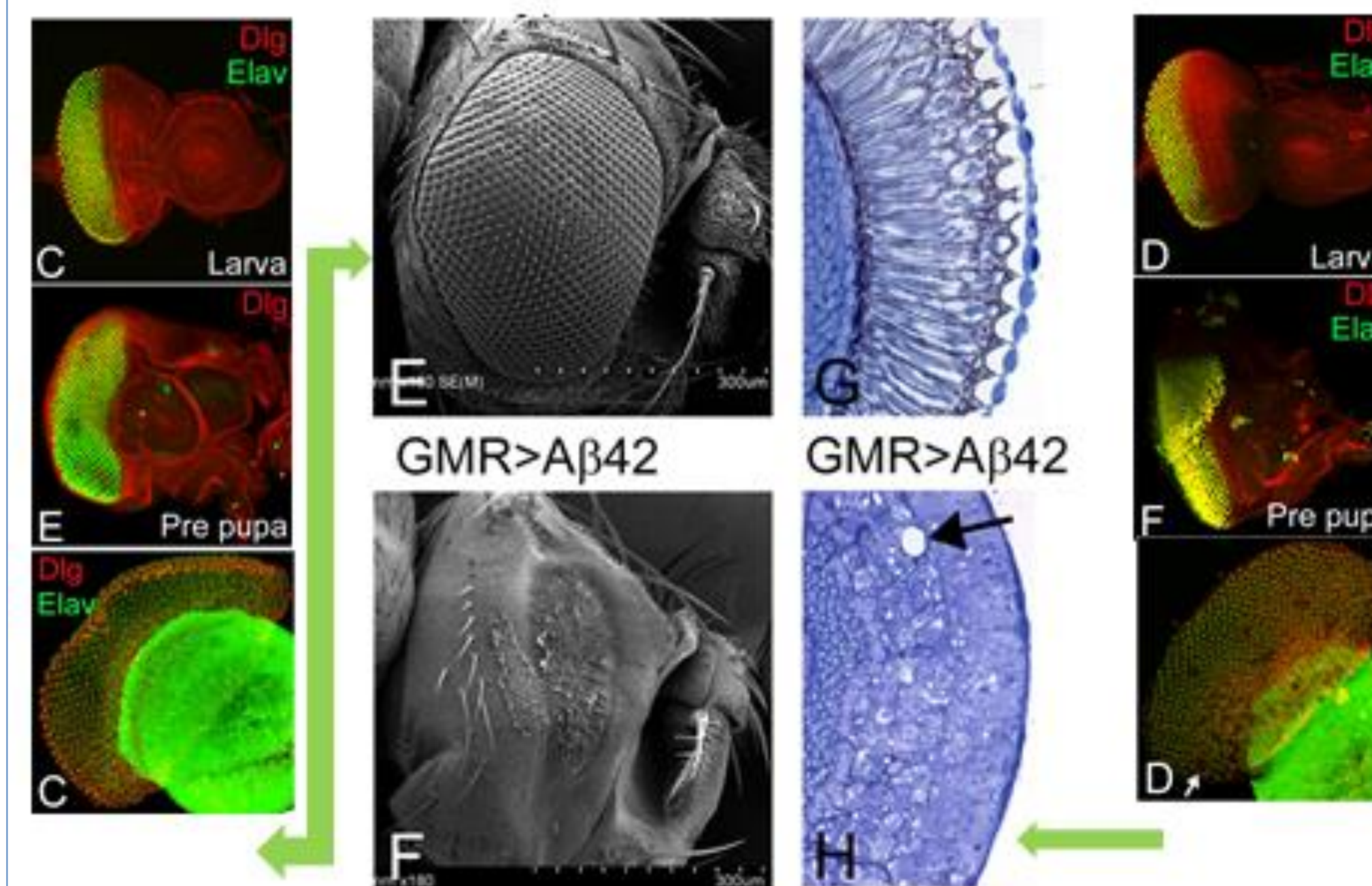


Alzheimer's Disease

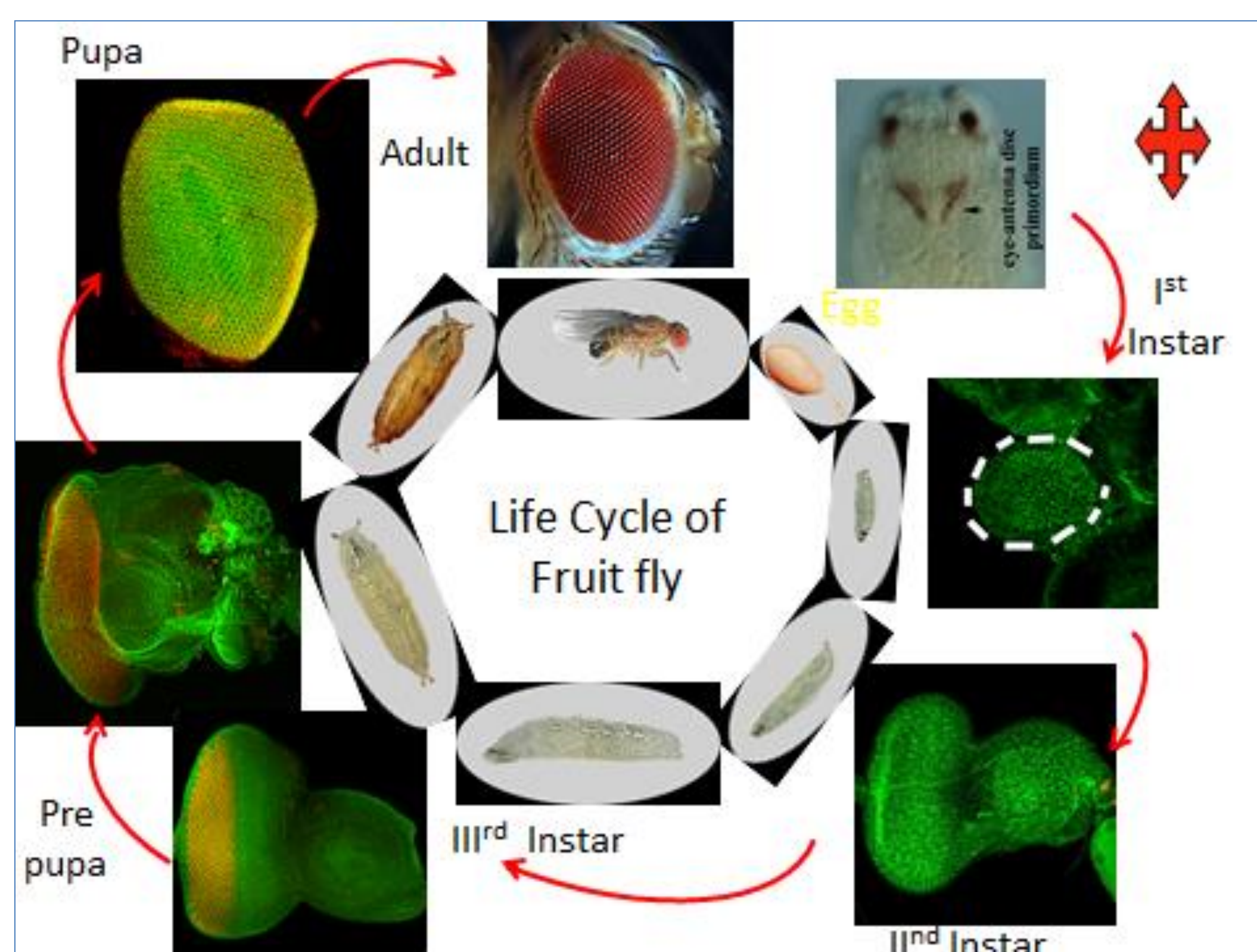
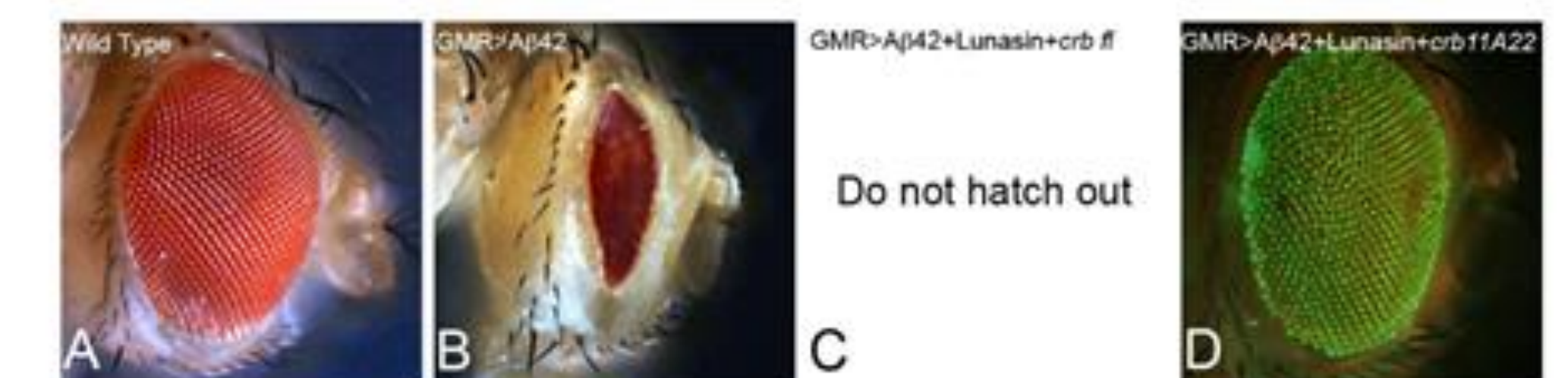
1. Amyloid Plaques
2. Neurofibrillary Tangles
3. Oxidative Stress due to ROS
4. Neuronal loss (Secondary)
5. Genetic Basis of ApoE



Drosophila eye model to study A β 42 mediated neurodegeneration

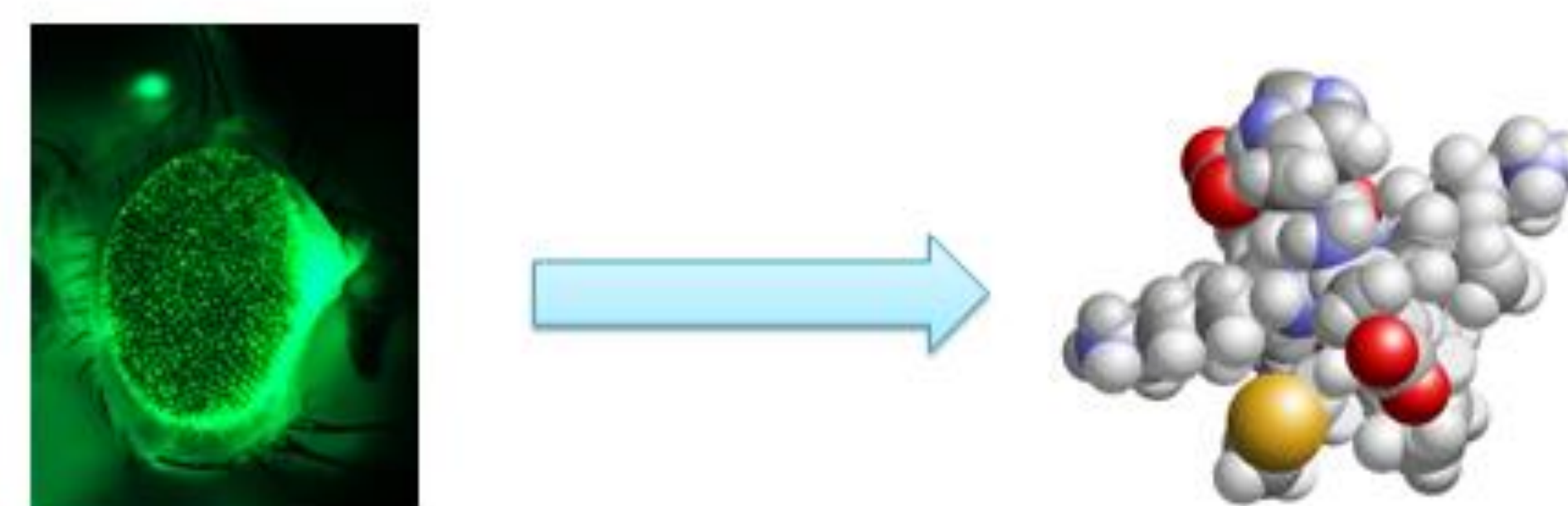


Downregulation of *crumbs* increases levels of Lunasin in A β 42 background



The Power of a soybean protein Lunasin

- Protein found within soy products
- 43 amino acids long
- Cancer research related to tumor suppression and decrease of cancer cell proliferation
- Heart disease research links Lunasin to be a anti inflammatory



Conclusions and Future Research

Conclusions

- In our preliminary findings we have seen that Lunasin is able to rescue the neurodegenerative A β 42 phenotype in our *Drosophila* eye model.
- The marker protein Wg expression is similar in our GMR>A β 42 flies and in our GMR>A β 42+Lunasin

Future Directions

- Discover the role Lunasin plays in regulating other protein markers (Crb) and pathways involved in A β 42 mediated neurodegeneration.
- Research the underlying effects of Lunasin that rescue the misexpression of A β 42 in the *Drosophila* eye model.